predictor for BDI score and physical and mental QOL scores. However, OH status independently predicted kidney disease-related QOL score ($\beta = -2.354$, $p = 0.012$).

Conclusion: In conclusion, OH negatively affects HRQOL in PD patients. Intensive efforts to control fluid overload may improve HRQOL of PD patients.

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Endovascular Salvage of Immature Hemodialysis Arteriovenous Fistulae

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Purpose: To assess the anatomical causes of immature hemodialysis arteriovenous fistula (AVF) and the outcome of the endovascular salvage.

Methods: Over a 4-year period, 51 dysfunctional and 3 thrombosed immature AVF were treated by endovascular intervention, which included percutaneous transluminal angioplasty and accessory vein obliteration by coil insertion or surgical ligation. Anatomical causes, clinical characteristics and the success rate of the endovascular salvage of the immature AVF were retrospectively analyzed.

Results: The access types were 27 radiocephalic fistulae, 25 brachiocephalic fistulae, and 2 transposed basilic vein fistulae. Mean interval from access creation to referral to angiography was 116 days (44–349 days). Anatomic problems were identified in 53 cases (98%). The causes of the immature AVF were stenosis (59%), accessory vein (22%), and combined stenosis and accessory vein (15%) and deeply located vein (4%) in the upper arm fistulae, and stenosis (67%), accessory vein (11%), combined stenosis and accessory vein (11%), poor surgical technique (7%), and deeply located vein (4%) in the forearm fistulae. New fistulae were created in the cases of immature AVF due to poor surgical technique, and surgical superficialization was done in the cases of deeply located veins. In the remaining cases, overall clinical success rate of endovascular salvage was 94.4%.

Conclusion: Immature AVF should be timely treated, because the most common causes are stenosis and accessory vein. Endovascular intervention can treat majority of cases with high success rate.

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Ethanol Induced Urine Acidification is Related with Early Acetaldehyde Concentration

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Background: Ethanol is worldwide favored beverage among the young adults, but heavy drinking cause many social problems and medical problems such as liver cirrhosis, central nervous system problem. These toxic effects are mainly caused by acetaldehyde, the metabolyte of ethanol. Usual dose of ethanol consumption can cause dehydration and mild metabolic acidosis, there is few experimental human data that explain about the association of blood ethanol and acetaldehyde level and aciduria. We investigated urine electrolyte and pH changes after moderate amount of ethanol ingestion in healthy young men for urine acidification change by ethanol and acetaldehyde level.

Materials and Methods: 30 Healthy young male volunteers were enrolled with informed consent. They ingested 1.3 g/kg of ethanol in fasting state, and blood and urine samples were taken at before ingestion, 1, 3, 5, 7 hours after ethanol ingestion. Snack was supplied after 4 hours of alcohol intake, and 500 mL of free water was allowed. Urine pH was measured by pH meter and blood and urine electrolytes were measured by indirect method. Blood ethanol was measured by enzymatic method and acetaldehyde was measured by spectrophotometer assay.

Results: 30 Volunteers were all males, age was 27.40 ± 2.7 years old, body weight was 72.83 ± 7.9 kg. Their usual limit of alcohol intake by history taking were 78.35 ± 56.7 gram, and average amount of ingested ethanol was 98.63 ± 10.7 gram. Blood ethanol concentrations were 0.131, 0.097, 0.098, 0.027% in 1 hr, 3 hrs, 5 hrs, 7 hrs after drinking respectively, and blood acetaldehyde concentrations were 1.480, 1.734, 1.221, 1.462% in 1 hr, 3 hrs, 5 hrs, 7 hrs after drinking respectively. Urine pH were 6.056 in before ethanol ingestion, and 5.724, 5.598 each of 3 hours and 7 hours after ethanol ingestion, respectively. Post-alcohol ingestion urine pH was significantly decreased ($p = 0.002$). Urine sodium and chloride excretions were not changed after ethanol, but urinary potassium excretion was significantly decreased (61.09 ± 25.3 to 40.16 ± 11.9, $p < 0.001$). There was no correlation between urine pH and urine electrolyte excretion. Interestingly, ealrly elevation of serum aldhehyde (1 hour after ethanol) and early urine acidification (3 hrs after ethanol) showed strong correlation ($r2 = 0.189$, $p = 0.021$), and after 7 hrs of ingestion, urine pH was increased ($r2 = 0.383$, $p < 0.001$). There was no gastrointestinal trouble, neurological problem, and other ethanol induced medical problem.

Conclusion: In conclusion, urine acidification after ethanol ingestion is related with serum acetaldehyde concentration. Early elevation of acetaldhyde could induce urine acidification, but the urine pH was elevated after a few hours, that might make prolonged acidemia.

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Dipeptidyl Peptidase IV Inhibitor MK-0626 Attenuates Pancreatic Islet Injury in Tacrolimus-Induced Diabetic Rats

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Background: Tacrolimus (TAC)-induced pancreatic islet injury is one of the important causes of new onset of diabetes in transplant recipients. This study was performed to evaluate

whether a dipeptidyl peptidase (DPP) IV inhibitor is effective in improving TAC-induced diabetes mellitus by reducing pancreatic islet injury.

Methods: Rats were treated with TAC (1.5 mg/kg, subcutaneously) and DPP IV inhibitor MK-0626 (10 or 20 mg/kg, oral gavage) for 4 weeks. The effect of MK-0626 on TAC-induced diabetes was evaluated by assessing pancreatic islet function, histopathology. TAC-induced incretin dysfunction was also examined with the serum active glucagon like peptide (GLP)-1 level after glucose loading. Protective effect of MK-0626 was evaluated by measuring markers for oxidative stress, oxidative resistance, and apoptosis. To reveal whether enhanced GLP-1 signaling is associated with these protective effects, we measured the expression of GLP-1 receptor (GLP-1R) and effect of GLP-1 analogue exendin-4 on cell viability and oxidative stress in isolated islets.

Results: MK-0626 treatment attenuated TAC-induced pancreatic islet dysfunctions and islet morphology. TAC treatment showed defective in active GLP-1 secretion; however, MK-0626 recovered these effects. TAC treatment increased the level of 8-hydroxy-2'deoxyguanosine (8-OHdG), number of apoptotic cell death, active caspase-3 and decreased the level of manganese superoxide dismutase and heme oxygenase-1, and MK-0626 treatment reversed these changes. MK-0626 treatment restored the expression of GLP-1R and direct exendin-4 treatment in isolated islet reduced TAC-induced cell death and 8-OHdG expression.

Conclusions: DPP IV inhibitor MK-0626 is an effective antidiabetic agent with antioxidative and antiapoptotic properties by enhanced GLP-1 signaling in TAC-induced diabetics. These beneficial effects of DPP IV inhibitor could be helpful to a delay in the new onset of diabetes in transplant recipients.

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Effect of Blocking with Minicircle DNA of Interleukin-6 on Skin Allograft Rejection

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Blocking of proinflammatory cytokine, interleukin-6 (IL-6) is effective in decreasing resistance to allogenic tolerance. In this study, we investigated whether anti-IL-6 receptor producing nonviral minicircle (MC)-DNA, is competent to inhibit alloresponse-induced IL-6 in highly immunogenic murine skin allograft model. We designed MC-DNA producing IL-6R antibody and verified in vitro system. One day before skin allograft modeling, systemic MC-DNA exposed via hydrodynamic delivery of MC-DNA in vivo (50 ug of DNA, tail vein, single injection). Using GFP tagging MC-DNA, we confirmed its organ distribution. At day 5, we measured the amount of IL-6 and IL-6R antibody in serum. As functional examinations, we evaluated survival rate, morphological changes of graft, immune cell infiltration, and population of T helper 17 cells (Th17, FACS marker: CD4+/RoRγt) and regulatory T cells (Treg, FACS marker: CD4+ Foxp3+). We compared its alloimmunity and graft survival efficiency with the cyclosporine A (CsA)-treated group (50 mg/kg, daily administration via oral gavage). Hydrodynamic delivery of MC-DNA was mainly localized in hepatocytes. Serum IL-6 and IL-6R antibody detected in anti-IL-6R MC-DNA treated mice. At day 8.5, untreated mice completely rejected the graft confirming by daily observation of loss of skin graft and erosion. However, mice received either anti-IL-6R MC-DNA or CsA presented prolonged acceptance of graft until day 15 or 15.6, respectively. Results from morphological changes and immune cell infiltration in the graft were also consistent with survival rate. FACS results showed that anti-IL-6R MC-DNA treatment markedly suppressed Th17 population compared with the untreated mice. However, there was no effect on Treg population. On the other hand, administration of CsA showed the increased Th17 and decreased Treg population compared with untreated group. We found that single injection of nonviral minicircle DNA targeting IL-6R is effective in both acute allograft acceptance and allogenic tolerance. This suggests that simple and effective gene therapy method using antibody producing minicircle DNA may represent a powerful tool for the transplantation.

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